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THE REACTIONS OF A HETEROGENEOUS POPULATION TO PERTURBATION. An interpretative model of mortality trends in Russia♦

On the eve of the Second World War, the USSR was lagging far behind the rest of the developed world with regard to life expectancy: 16 years behind France, for instance. This gap was virtually filled in during the period 1946-60, but the progress marked time in the early 1960s and then gave way to a slow erosion.

The mortality decline resumed during the 1980s, with a sharp acceleration in 1985. In the space of three years, male life expectancy increased by three years, as though for each year that had been lived the slate was wiped clean and the number left to live was unaltered. In 1991, however, there was a spectacular new turnaround: again in the space of three years, not only was all the previous gain lost, but men's life expectancy was cut back by six years. The end of the restrictions on alcohol sales, and the economic disorganisation brought about by the change of political regime, have been blamed for this sharp regression. In the present study, A. AVDEEV*, A. BLUM**, S. ZAKHAROV*** *and* E. ANDREEV**** *suggest another possibility, namely that it is largely, or at least in part, the result of a return from the exceptionally favourable period 1985-90 – when the population most at risk of dying from a number of causes was temporarily spared – to the evolution observed in previous decades. The reversal in 1991-95 was impressive, and the mortality trends continue to be unfavourable today.*

The present article proposes another methodological approach and a somewhat different interpretation of Russian mortality from the studies published in Population *in recent years, in particular issues 1989-6, 1994-2, 1995-4/5 and 1996-3 (*Population: An English Selection*, 7-1995, 8-1996 and 9-1997).*

Demographers have often been preoccupied with the effects of heterogeneity on measures of the principal parameters of population dynamics. As early as 1878, Lexis criticized Quetelet's 'average man' on these grounds (Lexis, 1878), and the controversy was to last into the 20th century. Demo-

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graphy served to give concrete expression to the criticisms that were levelled at Quetelet's approach. In developing the principle of standard populations, this science privileged an approach that defined *a priori* the relevant criteria of heterogeneity, and analysis was founded on indicators designed to eliminate this factor. Thus, the structure of a population was considered as a sum of sub-populations with homogeneous behaviours, these behaviours being defined by average quantities.

Some studies have, however, investigated how a heterogeneous population evolves and changes (Vaupel, Manton and Stallard, 1979; Pollard, 1993), or have used this criterion to suggest a possible interpretation of certain dynamics (Yashin *et al.*, 1985; Andreev, 1995b). But such studies have remained largely theoretical and have not resulted in any practical applications.

The notion of 'disturbance' is also well known in demography. In historical demography, the mechanism of demographic crises is founded on this concept, even if no general model has really explained the underlying individual models. In the framework of the general theory of population dynamics, the recovery of the stable state after a catastrophe has been studied by several authors (Le Bras, 1969; Blanchet, 1988). These formalizations reflect how a disturbance affects the stable population structure, and the theorems of convergence towards stability have thrown light on the consequences of exogenous shocks, or of modifications due to gradual behavioural changes in one or several sub-populations (Bourgeois-Pichat, 1994).

This notion is often linked to that of 'catching-up' process, defined in terms of timing changes to explain fluctuations in fertility. It is not appropriate, however, in the case of mortality: a birth that has been postponed may or may not be made up later, but a death that has been delayed will always occur ultimately. The distinction between renewable and non-renewable events thus calls for a different formalization in each case.

The Russian mortality trends observed during the 1980s and 1990s provide an excellent example of how a heterogeneous population may react to the momentary effect of an exogenous factor. The association of two approaches which, although classic, are rarely used, will provide a key for the (albeit partial) understanding of trends whose interpretation has given rise to considerable controversy.

I. – The context

No developed country, or even any developing country apart from in times of crisis, ever experienced such sharp mortality fluctuations within such a short space of time as Russia has (Figure 1 and Table 1). Deaths began to fall in the summer of 1985. Despite a slight rise at the beginning of that year owing to a flu epidemic, the urban standardised death rate fell by 1.5% for men and 6.4% for women compared to 1984, with a slighter reduction in the countryside: 0.3% and 5% respectively. Again compared

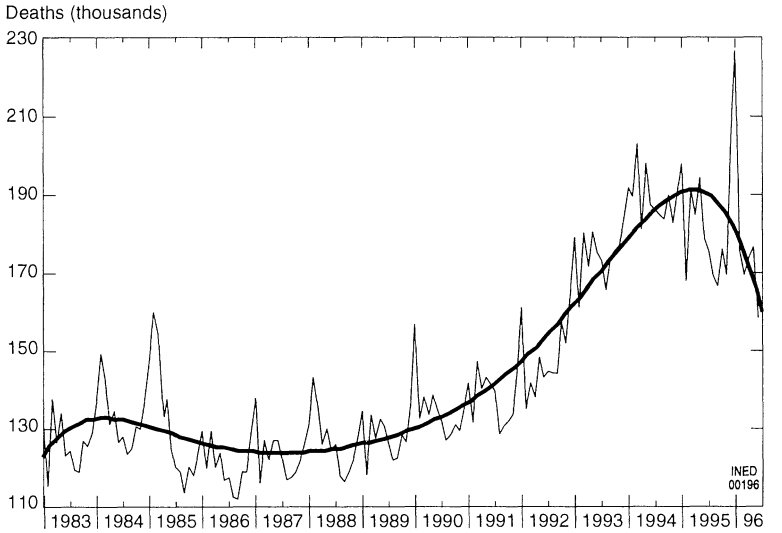

Figure 1. – Monthly numbers of deaths at ages 1 and over in Russia, January 1983 to July 1996 (crude and smoothed data)

N.B. : The data for 1996 are provisional

to 1984, the urban rate fell by 7.4% for men and 17.6% for women in 1986, and the rural rate by 8.9% and 18.3%. In the space of these two years, male life expectancy, which had been oscillating around 62.5 in the urban and 59.5 in the rural sector since the beginning of the decade, suddenly gained 2.8 and 3.9 years respectively. At the same time, the gender gap in mortality, which had been 10.5 years in the towns and 13.5 in the countryside, fell by 1.7 and 2.8 years respectively (Table 1).

The upturn in mortality which followed, from 1987 to 1994, was considerable. The number of deaths soared from 1.5 million in 1986 to 2.3 million in 1994. Male life expectancy, which in 1986-87 was already nothing to boast about for a developed country (64.9), lost 7.6 years within seven years, falling to a level of 57.3 in 1994 (Figure 1 and Table 1). The gender gap widened again, and in 1994 women had almost 14 years more life expectancy than men, a situation which was unprecedented in Russia or anywhere else in Europe[1].

This deterioration has been interrupted during the last two years, and life expectancy is once more increasing. But such a striking pattern, the like of which has never been seen in peacetime, raises many questions. Does it reflect the degraded social and health conditions that have accompanied the recent political upheavals? Is it, as has been suggested, the result of a deteriorating ecological context? What does it signify?

[1] Excepting the war years.

TABLE 1. – DEMOGRAPHIC INDICATORS FOR RUSSIA, 1979-95

Year	Crude death rate (per 1,000)		Infant mortality rate (per 1,000 live births)				Life expectancy at birth			
	Urban population	Rural population	Urban population		Rural population		Urban population		Rural population	
			Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls
1979	9.79	13.12	24.33	17.96	28.57	21.91	62.34	73.11	59.36	72.38
1980	9.99	13.39	24.25	17.72	26.89	21.06	62.20	73.07	59.33	72.29
1981	9.95	13.29	23.35	16.94	27.10	20.88	62.48	73.30	59.42	72.52
1982	9.79	13.02	22.18	16.25	24.97	19.05	63.03	73.77	60.24	73.04
1983	10.10	13.50	21.59	16.00	24.84	19.00	63.05	73.56	59.92	72.85
1984	10.56	14.25	22.86	16.84	26.25	20.67	62.55	73.16	59.40	72.16
1985	10.33	13.96	22.79	16.75	26.03	19.51	63.51	73.45	60.60	72.45
1986	9.58	12.46	21.40	15.76	22.83	17.32	65.30	74.28	63.45	73.92
1987	9.72	12.66	21.63	15.57	23.85	17.98	65.39	74.33	63.31	73.88
1988	9.86	12.96	21.18	15.39	23.28	17.94	65.22	74.33	62.82	73.72
1989	10.00	12.74	20.16	14.65	21.77	16.60	64.73	74.47	62.55	74.15
1990	10.41	13.29	19.73	14.51	21.31	15.58	64.56	74.27	61.88	73.84
1991	10.65	13.42	19.93	14.84	22.25	16.27	64.20	74.18	61.53	73.67
1992	11.48	14.03	20.42	15.06	22.27	16.37	62.37	73.70	60.57	73.30
1993	13.69	16.09	21.82	16.81	24.81	18.79	59.17	71.93	57.99	71.56
1994	14.90	17.19	20.32	15.06	22.97	17.00	57.77	71.23	56.93	70.92
1995	14.36	16.28	19.68	14.85	22.49	17.11	58.39	71.67	57.79	71.55

Sources: A. AVDEEV and A. MONNIER, *Mouvement de la population de la Russie 1959-1994 : Tableaux démographiques*, INED, Série *Données Statistiques*, Paris, 1996 ; and for 1995, Goskomstat RF, 1996.

The existence of a direct one-to-one relationship between the political changes and the strong variations in mortality during recent years has lately been challenged in a number of articles. They have privileged rather an interaction between these upheavals and a long-term trend that began in the early 1960s, and that had already diverted Russia from the path followed by most western countries (Blum *et al.*, 1994; Blum, 1994; Meslé *et al.*, 1996; Meslé and Shkolnikov, 1995; Avdeev and Monnier, 1996). The object of the present study is to explore in more depth the relationship between the sudden sharp turn-around of recent years and the long-term mortality trend.

A closer look at the age-specific mortality patterns suggested that cohort mortality trends should be investigated to discern the links between past evolutions and present ones, between long-term dynamics and short-term changes. The observation of cohort survivorship functions will reinforce the hypothesis that a 'catching-up' or 'compensation' process exists. We shall then elaborate the significance of such a concept with regard to mortality by introducing a factor of population heterogeneity.

The theoretical argument underlying our demonstration will then be expressed by an extremely simple model involving two separate sub-populations,

each one having a homogeneous behaviour but different from the other. This will reveal the type of effect such heterogeneity may produce, and allow us to measure it; the simplified form of the model does not, however, preclude other possible estimations.

The model will lead us to conclude that Russia's observed mortality trend can be explained to a large extent by supposing the risk of dying in both sub-populations has continued a long-term deterioration that only appeared to come to a halt in 1985. Thus, the recent turn-around merely reflects a resumption of this long-term process, and is not evidence of a rapid regression of social and health conditions during the last five years.

We consequently reconsider a number of inconsistencies observed in the recent mortality dynamics. We underline the very unusual pattern of mortality change in Russia since the early 1960s, in which we find strong grounds to explain the population's present sensitivity to the slightest disorders.

II. – A cohort issue?

A sharp decline of mortality between 1980 and 1987, and especially in 1986 and 1987, was followed by a strong rise between 1991 and 1995, and especially in 1993 and 1994. In other words, two opposite trends are observed in Russian mortality a few years apart, and of an amplitude that has probably never before been experienced in peacetime, in the absence of any deadly famine or epidemic. But these opposite movements are not symmetrical in terms of age (Figures 2 and 3), particularly for men: the mortality rise between 1980 and 1994 is highest around age 50, while the largest reduction between 1980 and 1987 was around age 40[(2)]. Thus, there has been a shift towards the older age groups, which might suggest that the downturn and the upturn are not entirely elements of one single process.

That directs us to cohort analysis, since these observations suggest that the cohorts the most concerned by the mortality decline are those that are now most concerned by the increase. Although this is not shown clearly by Figures 2 and 3, the hypothesis of a relationship in terms of cohorts, and not simply ages, merits further investigation.

We shall now, therefore, turn to cohort mortality analysis and the anomalies that this reveals[(3)].

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(2) To measure the mortality rise, we purposely chose 1980 instead of 1987 as reference year. The issue being the very rapid variations in mortality level, the minimum observed in 1987 may be just as problematic as the maximum in 1994. It is therefore preferable to take an ordinary year as basis for comparison.

(3) We note some other interesting features in Figures 2 and 3. The peak observed for men around age 18 certainly reflects the excess mortality due to military service and to duty in the regional conflicts that have broken out in Russia. It may also show a sharp rise in road deaths around the age when driving becomes legal. Finally, there may be underenumeration of the male population of these ages owing to military service. The rapid increase in female mortality at ages 15 and above between 1980 and 1994 may express a rise in maternal mortality and in deaths from complications of abortion among very young women. It should perhaps also be related to the road deaths hypothesis for men (of slightly older ages), since young women could be affected as passengers.

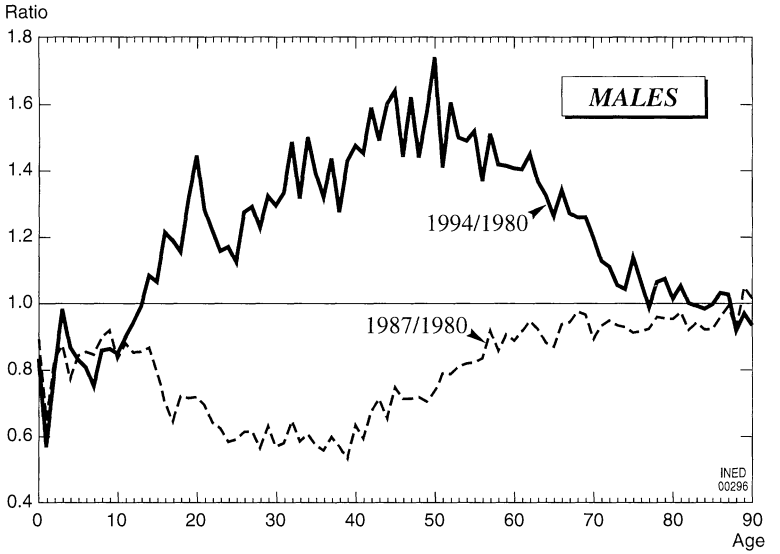


Figure 2. – Mortality change between 1980 and 1987 and between 1980 and 1994, males (ratio of crude death rates)

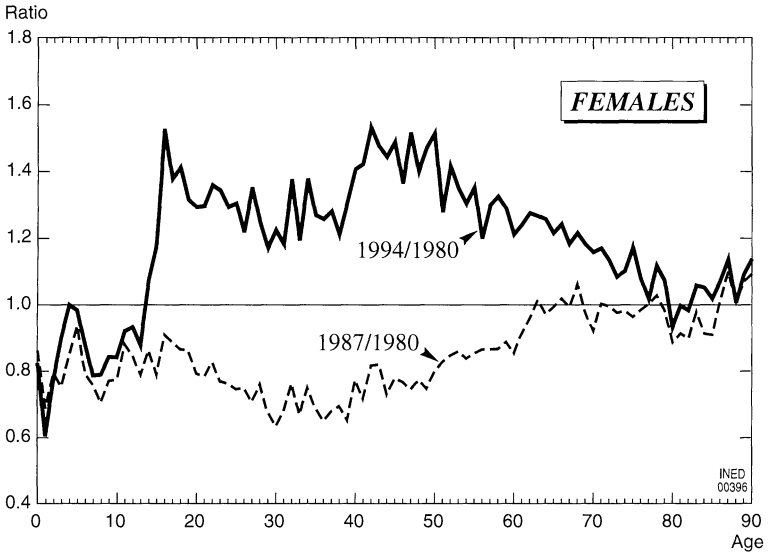


Figure 3. – Mortality change between 1980 and 1987 and between 1980 and 1994, females (ratio of crude death rates)

Cohort survivorship To calculate cohort survivorship probabilities, it was necessary to recompute the deaths by age and cohort from the deaths registered each year[4]. A smoothing and interpolation procedure was used (Bonneuil, 1994). Without going into details (these are provided in the Appendix), the method consists in interpolating surfaces of deaths cumulated by age and year of occurrence, on the basis of grids in which each year and age are divided into 24 parts, with application of a smoothing technique. We were then able to distribute the deaths by age and cohort, after which we could calculate the cohort probabilities of dying within a calendar year (1 January to 1 January). Thence we derived a survivorship function for each cohort on 1 January of each year, and thus at a given age each year.

Deaths and population by age were available only as from 1 January 1959, the year of the first Soviet census after the Second World War. The survivorship of cohorts born before 1959 could therefore not be reconstructed from the time of their birth, but only from the age reached during that year. We consequently reason henceforth in terms of *survivorship probabilities conditional on survivorship to certain ages*. From now on, we shall refer to 'survivorship in cohorts surviving at age 20' (or, more precisely, survivorship among those members of birth cohorts who have survived to the age of 20) denoted by the ratio $l(x)/l(20)$, where l is the survivorship function of a given birth cohort.

Survivorship in the cohorts surviving at age 20 can be calculated for birth cohorts 1938 to 1974, the former being aged 20 on 1 January 1959 and the latter on 1 January 1995. Earlier cohorts could not be used for lack of data and later cohorts were younger than 20 in 1995.

Birth cohort 1938 was aged 45 at the time when mortality started to fall dramatically. The survivorship curves of survivors to age 20 in this cohort and those born later (cohorts 1948 and 1958 were an arbitrary choice) perfectly synthesise mortality trends in the recent period, for men (Figure 4) and women (Figure 5) alike.

The curves for cohort 1938 and earlier (not shown in the figures, but provided in Adamets *et al.*, 1994) are regular and correspond to the classic survivorship functions in trouble-free times, apart from a very slight inflexion of the curve after age 45. In contrast, those of cohorts 1948 and 1958 both display a very clear inflexion at the age reached in 1986, 38 and 28 respectively. This reduces the slope of the survivorship function, then this is offset by an opposite trend in the early 1990s. The survivorship function then appears to regain the level it would have had without the

[4] The earliest data we have used are the Goskomstat series of deaths by sex and individual age for the year 1959; for that year, we have the population pyramid as of 1 January. From 1959 to 1970, no population pyramids were estimated; then, after the 1970 census, Goskomstat prepared population pyramids as of 1 January of each year, so that we were able to use the series up to 1 January 1996. For the years 1959 to 1970, we simply interpolated the populations of each age, given that this period is not directly the object of our study (we could alternatively have used deaths by age and births, but migration data by age are not well known and vary from year to year).

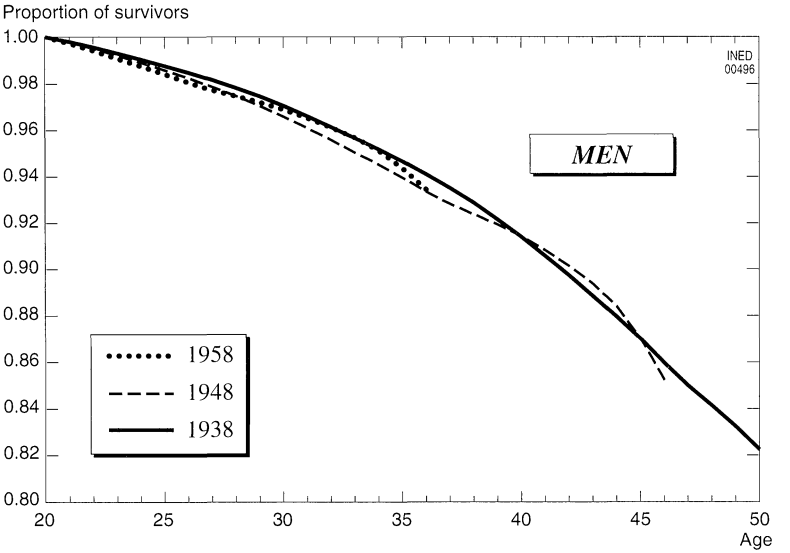


Figure 4. – Survivorship probabilities between the 20th and 50th birthdays among men born in 1938, 1948 and 1958 having survived to the age of 20

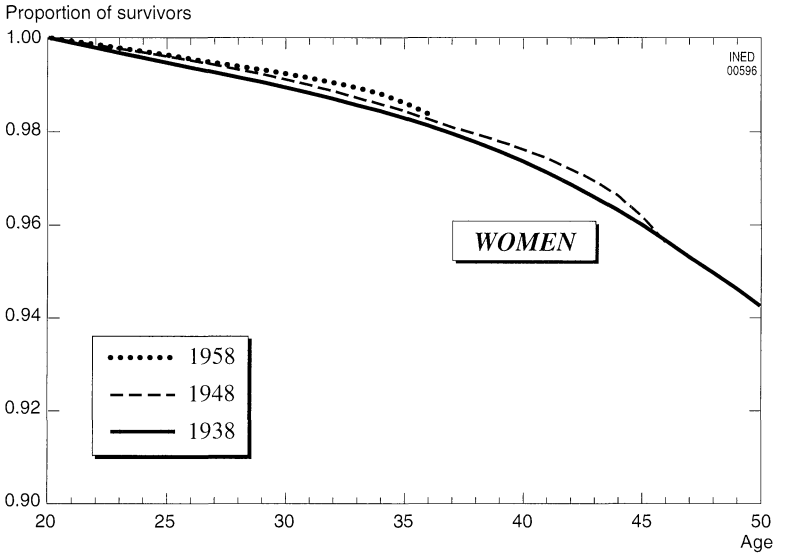


Figure 5. – Survivorship probabilities between the 20th and 50th birthdays among women born in 1938, 1948 and 1958 having survived to the age of 20

inflexion in 1986, although this 'compensation' calls for further investigation, which we now propose to do.

Our reconstruction clearly illustrates the complexity of the Russian mortality trends. In addition to these deviations, we note the consequences of the slow and continual deterioration since the beginning of the 1960s. Up to the mid-1980s, the survivorship probabilities of successive cohorts were consistently lower at all ages. The erosion was suddenly interrupted in the mid-1980s, but the current pattern replaces each cohort in this logic of progressive deterioration.

This first analysis suggests that the present increase of mortality resembles a catching-up process, in the wake of the fall observed in the mid-1980s. To confirm this, we estimated what pattern the survivorship functions would have followed in the absence of this deviation. We took the survivorship functions of the survivors to age 20 in cohort 1938 and the survivors to age 30 in cohort 1928 as standard[5]. We then used the Brass method to estimate the curves for survivors to age 20 (or age 30) in other cohorts, in the absence of any fluctuations. More precisely, we fitted the survivorship function of survivors to age 20 or 30 in a cohort g to that of survivors to age 20 or 30 in cohort 1938 or 1928, at the ages reached by cohort g up to 1984; thence we extrapolated the function in cohort g to the ages reached between 1984 and 1995 (see Appendix).

These extrapolations, illustrated by Figures 6 and 7 (male) and 8 and 9 (female) for the cohorts born in 1948 and 1958 studied earlier, show clearly that on 1 January 1995 the survivorship functions have drawn close to what they would have been had mortality not deviated. This is confirmed for all cohorts presently aged 20 and more (Table 2). Thus, if we follow the cohort trends, we note that the cumulated mortality as of today does not indicate any specially strong setback, but simply the logical continuation of a gradual deterioration that has been ongoing for the last 30 years. The observed cohort levels have simply reverted to those that would have been reached in the absence of the anomalies in the 1980s.

The current trends would thus represent a return to former trends, and express the transitory nature of the mortality decline in the early 1980s. There is no reason to state that the present mortality pattern is not merely a swing back from this substantial decline; there is no reason, therefore, to assert that it expresses a *recent* deterioration of health and social conditions.

To throw more light on the question, it is necessary to consider what the terms 'catching-up' or 'compensation for the decline', that we have

[5] The survivorship probabilities of cohort 1938 were used to extrapolate survivorship in cohort 1958, and those of cohort 1928 for cohort 1948. Indeed, cohort 1938 did experience a slight reduction of mortality, therefore a deviation of the survivorship curve, in 1981, at an age when we are attempting to extrapolate survivorship in cohort 1948. This explains the need to base the extrapolation on a cohort that did not experience these disturbances at the ages when cohort 1948 did. That is also why, for cohort 1948, we take survivors to age 30, since we do not have the survivors to age 20 in cohort 1928 (in any case, whether based on cohort 1928 or cohort 1938, the results obtained are similar).

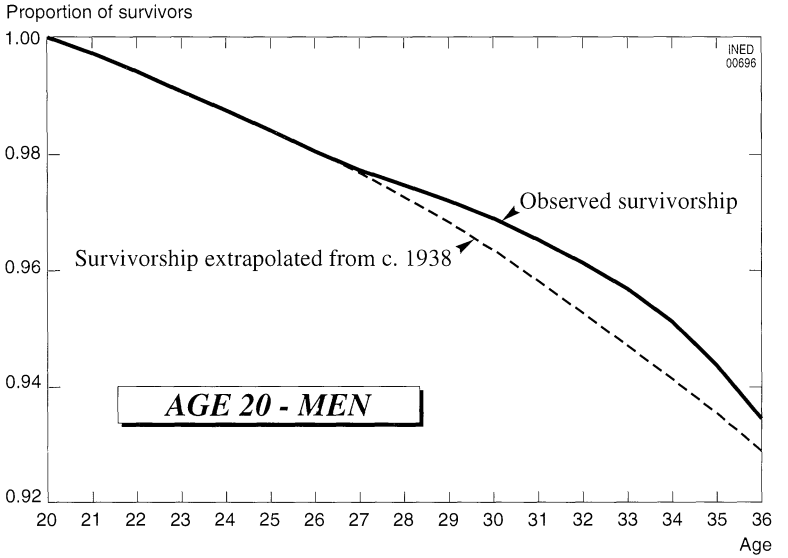


Figure 6. – Survivorship functions of men born in 1958 having survived to the age of 20, observed and extrapolated from cohort 1938

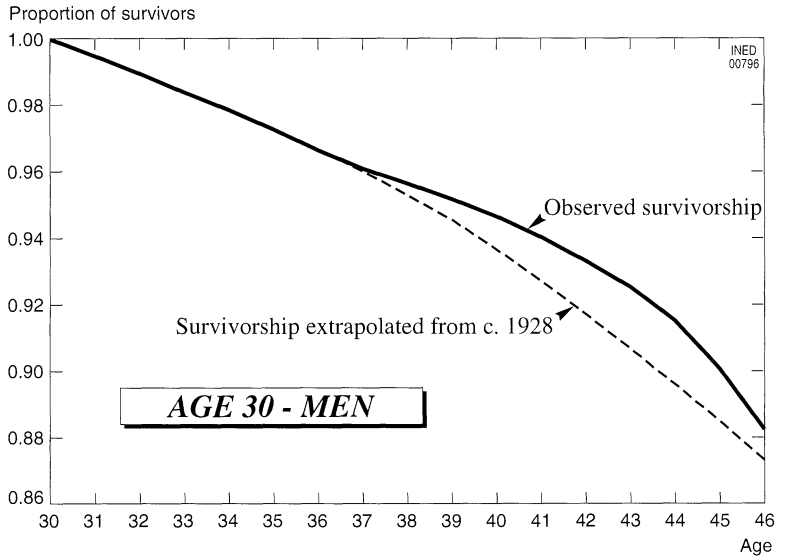


Figure 7. – Survivorship functions of men born in 1948 having survived to the age of 30, observed and extrapolated from cohort 1928

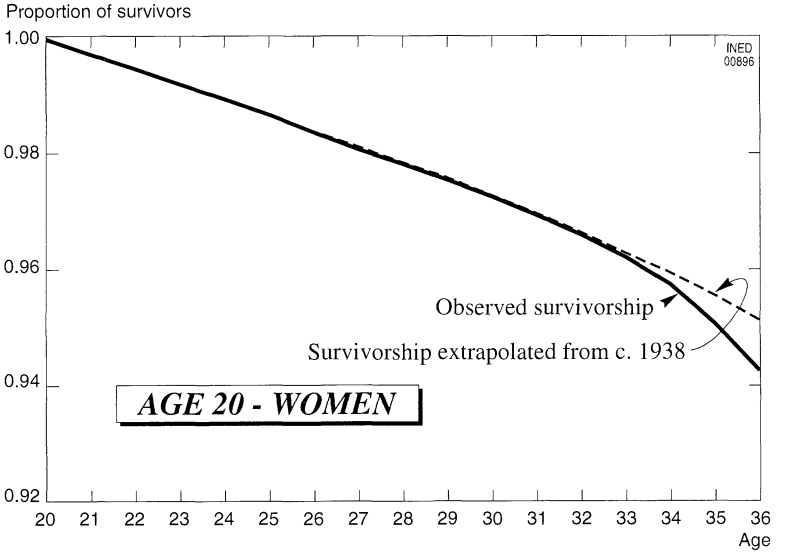


Figure 8. – Survivorship functions of women born in 1958 having survived to the age of 20, observed and extrapolated from cohort 1938

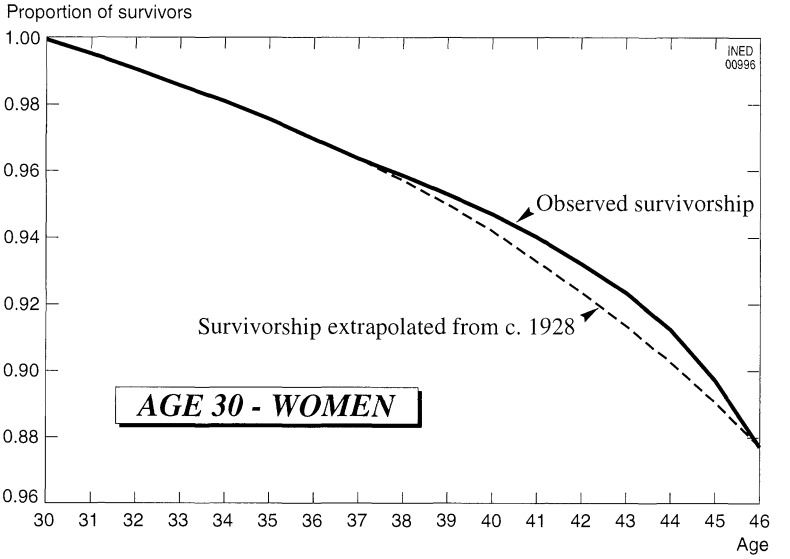


Figure 9. – Survivorship functions of women born in 1948 having survived to the age of 30, observed and extrapolated from cohort 1928

TABLE 2. – RATIO OF SURVIVORSHIP FUNCTIONS OF SURVIVORS AGED 20, SF OBSERVED IN COHORTS OVER SF FITTED TO COHORT 1938

Birth cohorts	Men	Women
1938	1.000	1.000
1939	1.005	1.004
1940	1.005	1.002
1941	1.006	1.003
1942	0.991	1.001
1943	1.002	1.001
1944	1.005	1.005
1945	0.996	1.001
1946	0.995	1.001
1947	0.994	1.000
1948	0.990	1.000
1949	0.988	1.000
1950	0.986	1.000
1951	0.984	1.000
1952	0.986	1.000
1953	0.991	1.002
1954	0.991	1.002
1955	0.991	1.002
1956	0.993	1.002
1957	0.995	1.002
1958	0.999	1.002
1959	1.003	1.003
1960	1.003	1.003
1961	1.004	1.002
1962	1.009	1.003
1963	1.015	1.003
1964	1.031	1.007

used here to describe survivorship trends, actually mean. In the field of fertility, the concept has long been debated, in particular with a focus on the tempo/quantum opposition and the relationships between period and cohort indicators. But this distinction is meaningless in the field of mortality: a birth can be delayed, then caught up or not, whereas a death can only be delayed.

We propose, therefore, to *introduce a factor of heterogeneity* in the population, using a model that we have simplified in the extreme. Like any model, it will serve only to guide us in our interpretation, and to follow through the potential consequences of any hypotheses made.

The assumption of heterogeneity We will assume that the Russian population is heterogeneous, and is made up of two sub-populations, one with a higher risk of dying than the other. We suppose the risk is higher in the second sub-population because of deaths related directly or indirectly to alcohol consumption, that is, a risk of the form $\mu_1 + \mu_2$, where μ_1 is the risk common to both sub-populations and μ_2 is the additional risk experienced by the second. As a result of the anti-

alcohol campaign and the restrictive measures introduced, the risk of dying falls in the high-risk group and remains stable in the other. Consequently, the number of deaths observed in the second sub-population is lower than it would have been without these measures, and its size increases at each age, compared to what it would have been otherwise. In the meantime, the first sub-population is not modified, since its mortality is not affected by the campaign.

After the withdrawal of these 'protective' measures, we suppose the risk of dying in the second sub-population returns to its earlier level. But the share of this high-risk group in the total population is larger that it would have been without the temporary downturn of mortality. Thus, the overall mortality risk also becomes higher than prior to the campaign. It will not return to its previous level until the composition of the population becomes stable again.

Thus, the total death rates may have risen beyond their earlier level, without having risen within each of the sub-populations. In this case, *the extent of the mortality rise is caused solely by a structural effect related to heterogeneity.*

The analysis of the cohort survivorship functions presented above is consistent with this model. The inflexion of the curve shows that a substantial number of deaths were averted by the measures introduced in 1986: these deaths can be attributed to the high-risk population. Now, the observed survivorship probabilities have been higher, so far, than they would have been without this inflexion: consequently, we can suppose that the additional deaths observed today correspond to deaths averted in the mid-1980s in the high-risk population, which then resumed its earlier mortality level.

Let us now formalize the model in order to evaluate the impact of such heterogeneity.

The model We propose here an extremely simple model. A slightly different formulation, allowing for more complex assumptions, in particular on the distributions of the two sub-populations and of the related mortality risks, is provided in Appendix 3.

Let us suppose that a factor, denoted Z, augments mortality in a segment of the population. Its temporary removal produces a sizeable drop in mortality, then when it begins to operate again, the overall mortality risk increases beyond its initial level.

The hypothesis

The population is made up of two sub-populations, and Z acts only on the second one, which we term the *high-risk group.*

The risk of dying is significantly higher in the second sub-population because of *Z*. If the action of *Z* is arrested for some lapse of time, the share of the high-risk group in the total population will increase. Mortality overall will consequently rise sharply when *Z* resumes its action.

The notations

t_0: point in time when *Z* stops acting;

T: duration of time throughout which *Z* is inactive. Action is resumed at time $t_0 + T$;

l(x): survivorship function of the whole population up to point t_0;

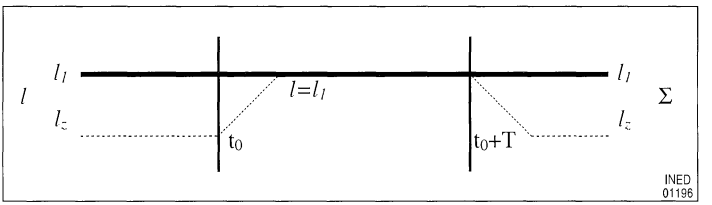
$l_1(x)$: survivorship function of the first sub-population; this is also equal to the survivorship function of the whole population at each date between t_0 and $t_0 + T$, the period when Z is inactive;

$l_2(x)$: survivorship function of the second sub-population (high-risk group) when Z is in action;

$\sum(x)$: survivorship function of the whole population after time $t_0 + T$;

$k(x)$: share of the second sub-population (high-risk group) in the total population of age *x* up to time t_0.

These notations can be summarized as follows:



Formalization of the hypothesis

Given $L(x) = l_2(x)/l_1(x)$ the ratio between the survivorship functions of the second and first sub-populations. Let us assume that, while *Z* is in action, this ratio is identical in all cohorts, and does not depend on $l_1(x)$. Then, in a stationary situation, that is until t_0, this ratio is equal to that calculated from cross-sectional survivorship functions.

Given $R(x) = l(x)/l_1(x)$ the ratio between the survivorship functions of the total population and the first sub-population. It is equal to the ratio between the survivorship function of the whole population until *Z* ceases to operate and after the effect of this factor has been totally eliminated.

In case of stability, that is, when $L(x)$ does not vary over time – and more specifically before time t_0 – the share of the second sub-population among the total population is described by a function $k(x)$ equal to:

$$k(x) = \frac{k(0)L(x)}{(1 - k(0) + k(0)L(x))} \quad [1]$$

Indeed, the number of members of each cohort surviving to age x is equal to:

$$l(x) = k(0)\, l_2(x) + (1 - k(0))\, l_1(x) \quad [2]$$

where the first term describes the dynamics of the second (high-risk) population and the second term the dynamics of the first sub-population. The share of the high-risk group is then:

$$k(x) = \frac{k(0)\, l_2(x)}{k(0)\, l_2(x) + (1 - k(0))\, l_1(x)}$$

By dividing the numerator and the denominator by $l_1(x)$, we obtain equation [1].

From [2] and dividing both terms by $l_1(x)$, we obtain:

$$R(x) = 1 - k(0) + k(0)\, L(x) \quad [3]$$

Finally, when there is a possibility of estimating $L(x)$ from available mortality statistics, the reciprocal of equation [3] is very useful:

$$L(x) = \frac{R(x) + k(0) - 1}{k(0)} \quad [4]$$

Thus, equation [4] displays the following important property:

Property no. 1: *Function L(x) is determined uniquely by R(x) and k(0)*

This property permits the direct estimation of $R(x)$.

Since the mortality level is higher before t_0 than after t_0, $R(x) = l(x)/l_1(x) < 1$. Thence, for $l(x) > 0$, $R(x) > 1 - k(0)$. This gives the following property:

Property no. 2: $1 - k(0) < R(x) < 1$ [5]

Inequality [5] has one of two possible consequences: either Z acts on a limited age range, or, if not, there is a limit strictly lower than 1 for the ratio $R(x) = \frac{l(x)}{l_1(x)}$ as x increases.

To perform the calculations, we assume that Z acts on ages $x < x_1$, and that its action starts from a given age x_0, that is, concerns the interval:

$$x_0 < x < x_1$$

We denote $k(t, x_0)$ the proportion of the second (high-risk) sub-population in the cohort aged x_0 at time t. We assume *this quantity is time invariant, both before and after* t_0, *and denote it* $k(x_0)$ *independent of* t.

At time $t_0 + T$, when Z resumes its action, the proportion of the first sub-population aged x, which we denote $k_1(x)$, is at ages $x > T$ equal to $k(x - T)$, since during the period T the whole-population mortality level has been the same as in the first sub-population. If $x - T$, then $k_1(x) = \mathrm{k}(x_0)$.

The survivorship function at time $t_0 + T + 1$, $l_1(x)$, can be defined by the probability of survivorship $p_1(x)$ between $t_0 + T$ and $t_0 + T + 1$, equal to:

$$p_1(x) = \frac{k_1(x)\, l_0\,(x+1)\, L(x+1)}{L(x)\, l_0\,(x)} + \frac{(1 - k_1(x))\, l_0\,(x+1)}{l_0\,(x)} \qquad [6]$$

The left-hand member describes the dynamics of the first sub-population and the right-hand member the second (high-risk) sub-population.

From [6], it is easy to see that, after a year, the proportion of the high-risk group aged x becomes equal to:

$$k_2(x) = \frac{k_1(x)\, L(x+1)}{k_1(x)\, L(x+1) + (1 - k_1(x))\, L(x)} \qquad [7]$$

Similar equations can be used to define $p_m(x)$ and $k_m(x)$, denoting respectively the survivorship probability and the share of the first sub-population within the total population, $m - 1$ years after time $t_0 + T$.

To measure the increase in this share, during the period when Z is not in action, variables $k_1(x)$ and $k(x)$ can be used. This ratio, under the condition $x > T$, is equal to:

$$\frac{k(x-T)}{k(x)} = \frac{R(x)}{R(x-T)} \cdot \frac{R(x-T) + k(x_0) - 1}{R(x) + k(x_0) - 1} \qquad [8]$$

and otherwise, from [1] and [4], to 1.

We note also that:

$$\frac{1 - k(x-T)}{k(x)} = \frac{R(x)}{R(x-T)} \qquad [9]$$

The derivative of [8] in relation to $k(x_0)$ is equal to:

$$\frac{R(x)}{R(x-T)} \cdot \frac{R(x) - R(x-T)}{((R(x) + k(x_0) - 1))^2} \qquad [10]$$

Given that $R(x)$ decreases with age (mortality at any age is no lower in the total population than in the second group, and so the differences automatically cumulate), the quantity [10] is lower than 0. Thence, *ceteris paribus*, the difference is all the greater as $k(x_0)$ is smaller. Thus, the maximum effect, that is, the maximum relative increase in the size of the high-

risk group (undergoing the action of Z), is attained when the value of $k(x_0)$ is minimum. We note that, from [5], we have $k(x_0) > 1 - R(x_1)$.

We can then formulate two further properties.

Property no. 3

Once $R(x_1)$ has been fixed, the impact of heterogeneity on the survivorship function is all the greater as the high-risk group is smaller and as the risk it is exposed to is large.

Property no. 4

Once $R(x_1)$ has been fixed, the relative size of the high-risk group is at least equal to $1 - R(x_1)$.

The above formal analysis now enables us to propose a set of numerical tests which interpret the model parameters in various ways.

Interpretation of the model parameters and numerical analysis

Four periods can clearly be distinguished in the mortality dynamics of Russian males 15-60 (Figure 10):

— from 1964 to 1981, the probability of dying between the 15th and 59th birthday ($_{44}q_{15}$) grows almost linearly;

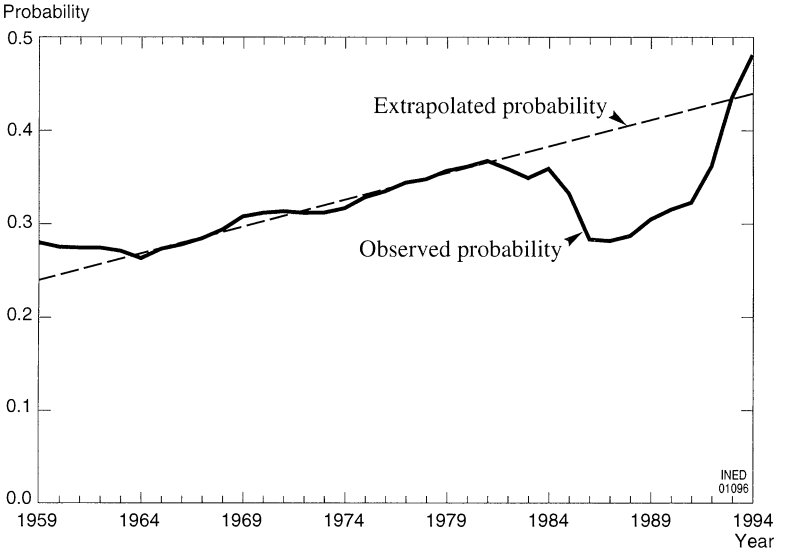


Figure 10. – Probabilities of dying between the 15th and 59th birthday, observed and extrapolated

— from 1981 to 1986, this probability falls, with an acceleration at the end of the period;

— from 1986 to 1991, there is relative stability and a slow growth, compared to the first period;

— from 1991 to 1994, the probability increases rapidly.

The model parameters can be defined as follows: $x_0 = 15$; $x_1 = 60$; $l(x)$ corresponds to the data for 1981 and $l_1(x)$ to the data for 1987 (we have used the data interpolated from the abbreviated life table, Russian males).

By attributing various values to k_0, we can estimate the $R(x)$ and $L(x)$ functions and calculate the other specifications, trying to come as close as possible to the survivorship function observed in 1994.

The most difficult parameter to estimate was T, since in fact the impact of Z has not changed suddenly. Figure 10 suggests that T should be set at 6 years, which corresponds to the period 1986-91.

With a minimum value of $k_0 = 0.12$, the $l_1(x)$ survivorship function appeared to be implausible. We therefore chose $k_0 = 0.14$ as scale for the cumulated effect.

The $R(x)$ and $L(x)$ functions are displayed in Figure 11 and $k(x)$ in Figure 12. Finally, the age-specific probabilities of dying observed in 1981, 1987 and 1994 are shown together with the results of the model in Figure 13.

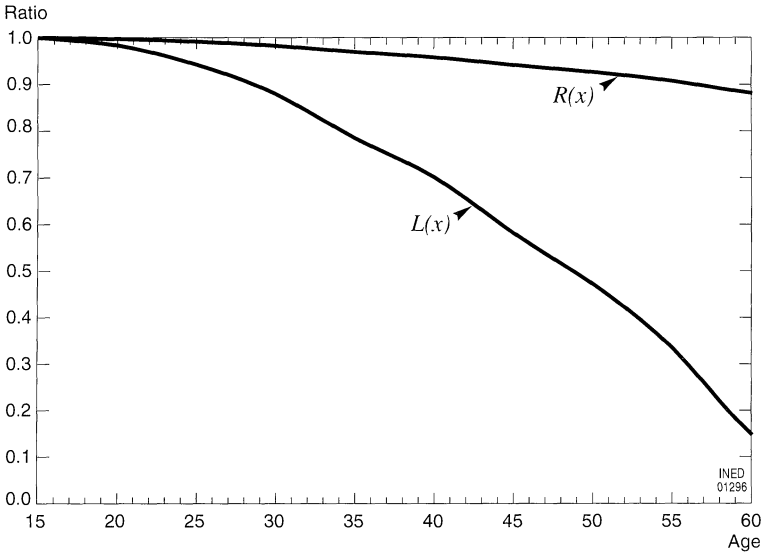


Figure 11. – R(x) and L(x) functions for T = 6, the model parameters being defined on the basis of 1981 and 1987

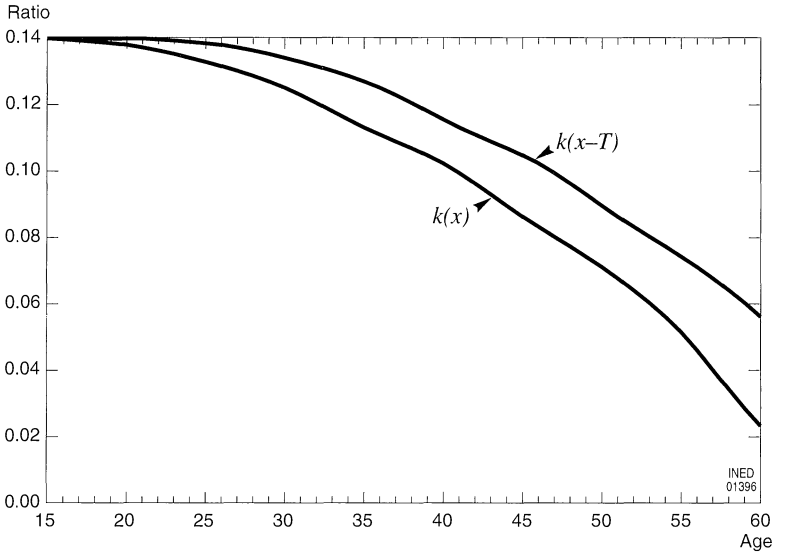


Figure 12. – $k(x)$ and $k(x-T)$ functions for $T = 6$, $k_0 = 0.14$, the model parameters being defined on the basis of 1981 and 1987

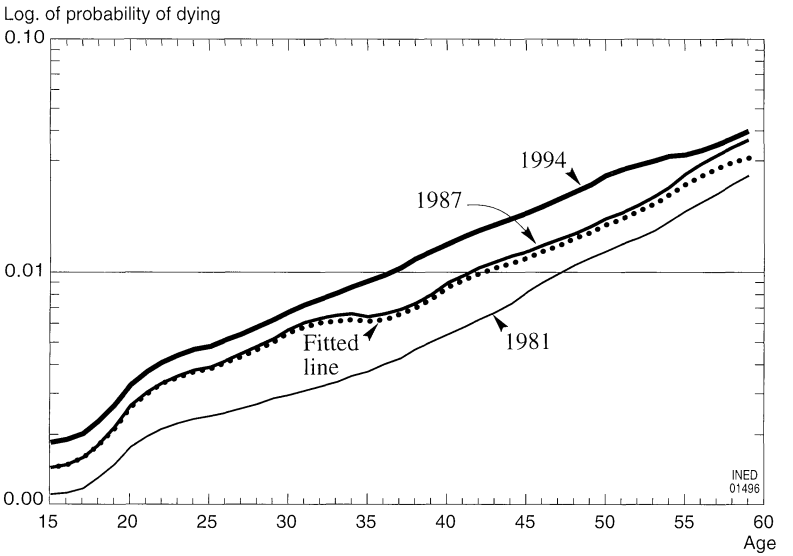


Figure 13. – Logarithm of the probabilities of dying in 1981, 1987 and 1994, observed and fitted using the model

The numerical impact of heterogeneity

The model enables us to obtain a number of important numerical results. The observed male probability of dying between the 15th and 59th birthday was 0.37 in 1981 and it had fallen to 0.28 by 1987. The model gave a probability of 0.39 in 1994, which is significantly higher than the observed probability for 1981, but even more significantly lower than that observed in 1994: 0.48. Thus taken as a working hypothesis, the effect explains only 21% of the mortality increase compared to 1981.

We note, however, that our model was constructed without taking into account the linear growth of mortality prior to 1981. For the ${}_{44}q_{15}$ probability, linear adjustment yields $R^2 = 0.97$ (that is, virtually a straight line); for the five-year probabilities of dying R^2 has an average value of 0.88 and a minimum of 0.70 in age group 25-29.

That allows a plausible extrapolation of the trend observed in 1964-1981 to the period 1982-92, and thus to estimate $R(x)$ as maximal value, during this period, of the ratio between the observed and the extrapolated survivorship functions. The maximum is not reached in 1987, but in 1988, and the curves have the best fit with $k_0 = 0.18$ and T again equal to 6.

Although the general form of the curves does not change, this second model yields results that are somewhat different from the first (Figure 14). The probability of dying obtained by extrapolation is 0.40 in the middle and 0.44 at the end of the period, and *the effect taken as working hypothesis in this case explains 46% of the rise in mortality between 1991 and 1994.*

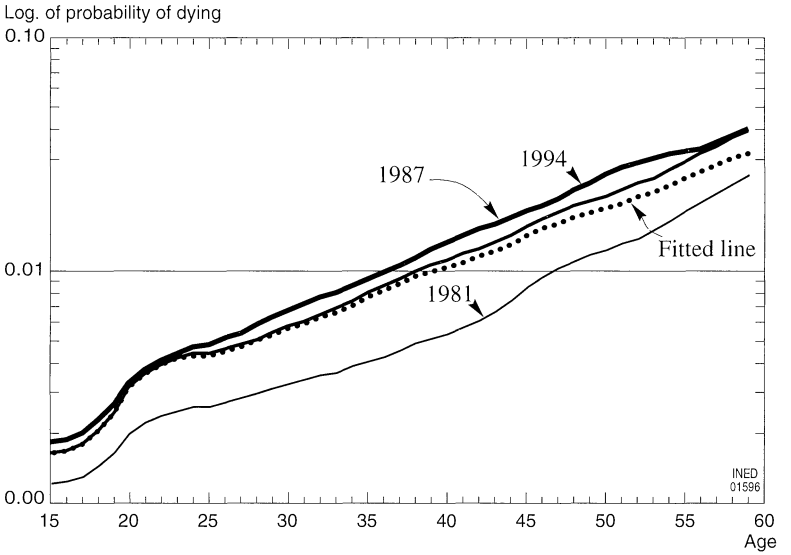


Figure 14. – Logarithm of the probabilities of dying in 1981, 1987 and 1994, observed and fitted using the second model

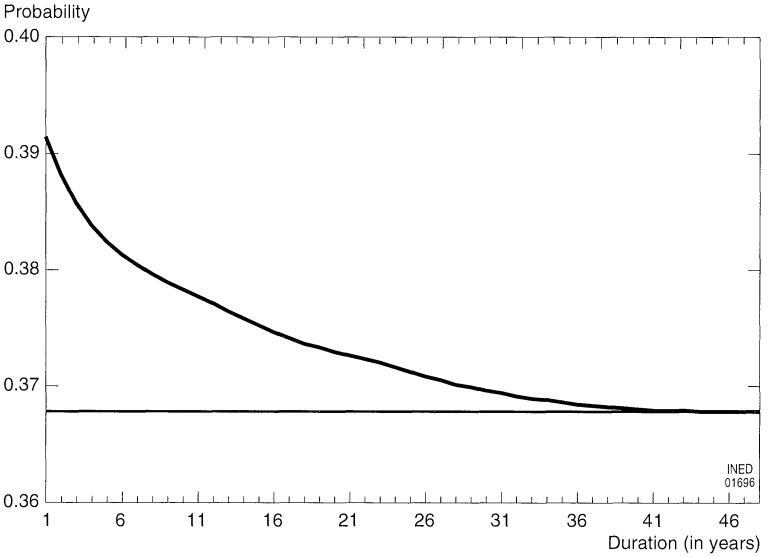


Figure 15. – Decrease in the probability of dying between the 15th and 59th birthday, after time t_0

Finally, the model suggests that the present exceptionally high mortality level will not hold for long. Because the share of the second subpopulation in the total population will decrease to its normal proportion, mortality will gradually return to the level in 1981 (Figure 15).

III. – Thirty-five strange years of mortality history

Explaining some inconsistencies

The approach we have proposed here provides a number of keys for interpreting some inconsistencies that have been noted in the Russian mortality trends. It also allows us to gain more understanding of the relationship between the atypical dynamics since 1960 and the sharp turnarounds of the last ten years or so.

The infant mortality rate fell in 1994 and 1995. True, it had risen by 10% in 1993; but this was a moderate rise, in fact, compared to earlier trends, all the more so since it probably included some artefactal growth due to the aligning of the index with international criteria (Andreev, 1995a). The present IMR is lower than in 1988. Yet this rate is generally considered to be closely linked to prevailing conditions, and to be an extremely useful indicator of the level of development[6]. It is particularly sensitive to the crises encountered by societies whose health system is not yet fully mas-

[6] See, among many others, « Manuel d'analyse de la mortalité », R. Pressat (ed.), WHO/INED, 1985, p. 47.

tered, which is the case of Russia (Andreev, 1995a). The growth of infant mortality during the 1970s had provoked many debates, and for several authors it was a definitive sign of the failure of the Soviet system. Why should the situation be any different today? It is sometimes argued that an infant health care system is easier to operate when there are fewer births per year. This seems a weak explanation, however, for the relatively favourable situation of recent years, when we consider that there was no rise in childhood mortality either between 1991 and 1995 (Figures 2 and 3). But if we follow our hypothesis, given that there was no mortality reduction at these ages in the mid-1980s, no catching-up effect was to be expected afterwards.

Similarly, the elderly have not experienced an upturn in mortality any more than the children. In fact, this upturn was limited to the population aged 20-60, who had previously benefited from the downturn in the mid-1980s. Were the recent deterioration due to a general erosion of the health care system, the elderly would certainly also have been affected.

The removal of these two inconsistencies points us back to the slow 35-year long evolution of Russian mortality. Indeed, it is obvious that such a strong reaction to exogenous events must also reflect an extreme sensitivity of the Russian people to voluntarist measures. This observation holds for the slow deterioration observed since 1960, which was only interrupted – temporarily – in 1981.

A new interpretation The above presentation thus led us to consider a long-term approach, the only one susceptible of explaining such rapid about-turns in mortality. The extreme sensitivity of the death rates to short-term disturbances is linked to the atypical nature of the long-term evolution that began in the 1960s, and which is now doubtless in its terminal stage.

Let us recall rapidly the Russian mortality trends which have been amply described elsewhere (Figure 16 summarizes them in life expectancies at birth). The mortality decline was interrupted in the mid-1960s and even gave way to a rise in both infant and adult mortality, particularly for males. This pattern is similar to those observed elsewhere in Europe: the mortality decline was interrupted just about everywhere at this time. In most countries, however, the hiccup was of short duration, and life expectancy quickly started to increase again and has continued to do so to the present day. The reasons for this process of interruption then recovery are now well known (Meslé, 1990; Meslé and Vallin, 1993, 1995; Caselli *et al.*, 1993; Caselli, 1993), and are basically related to the transition between two different mortality regimes.

The pattern was totally different in Russia. It is true that the growth of male life expectancy was arrested at roughly the same time as elsewhere, when Russia had practically caught up with the rest of Europe despite having lagged far behind before the war. Male life expectancy at birth was about 64 for men and 73 for women in 1960, not far behind France, with 67

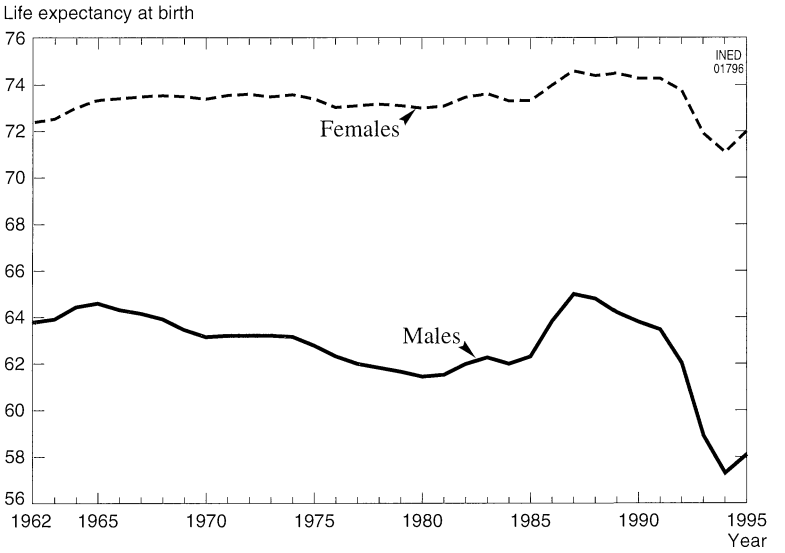


Figure 16. – Life expectancy at birth in Russia, 1961-94, males and females

and 73.5 respectively. But then, instead of resuming its progress, life expectancy in Russia continued steadily down; 15 years on, it amounted to 62 for men and 73 for women, while in France it had risen to 69 and 77.

A standstill in mortality can no doubt be explained by the need to implement changes in a health care system when the relative importance of infectious disease mortality dwindles (Blum and Monnier, 1989a, 1989b; Blum, 1992, 1994; Meslé and Shkolnikov, 1995). While mortality levels were high in Russia, up to the Second World War, the Soviet central planning policy maintained an effective health system by developing a highly technical form of medical surveillance based on immunization and the fight against infectious diseases. In the 1960s, however, when these diseases were coming under control, this technical conception of medical care was no longer adequate. It became necessary to convert to a policy of individual health care, regular check-ups by physicians, and preventive care using information campaigns. This was the path taken by western societies, but not by Russia, or Eastern Europe in general, a whole region which can be analysed along the same lines as Russia (Caselli, 1993; Labbé, 1995a, 1995b; Meslé, 1991).

However, if a standstill in mortality can be explained in this way, the slow deterioration observed in Russia between 1960 and 1980 necessarily involves another element. The principal victims were men aged 20-50 (Figure 17), which incriminates a factor specific to these ages. The blame cannot be laid on the erosion of the health system, since in this case childhood and old-age mortality would have risen most, and women would also have been

concerned. The age groups most affected are, in fact, those when hospital and medical care is sought least.

Here, we find the highly specific nature of Russian mortality: adults are particularly at risk, compared to other countries. There can therefore be no doubt as to the socio-economic aspect of this situation. Working-age adults are the most sensitive to acute social and economic tensions, an observation which is backed up by cause-of-death analysis (Meslé *et al.*, 1996): the 'Russian difference' lies essentially in the causes that reflect the relationship between man and his socio-economic environment, namely, violent deaths (suicides and accidents) and alcohol-related mortality. The excess mortality of men aged 20-60 could then express a growing reaction towards a working world in which they are involved but in which they do not really participate. Children and the elderly are too young or too old to feel the implications of this contradiction; they are sensitive only to the stagnation of the health system and to its inadequacies given the new mortality context.

Under these conditions, it is possible to interpret the halt to the mortality rise in the early 1980s as expressing the first signs of an opening of the social spectrum. The close relationship between society, economy and mortality then amplified the impact of the social changes, a fact which is clearly reflected in the sharp acceleration of the mortality decline in the mid-1980s. Russian society was moulting, and in this context the anti-alcohol campaign was one element among others: it more directly accelerated the trend, but was not the underlying motive (Figure 18).

The fight against alcoholism has, in fact, a long history and is marked by fundamental contradictions. A rise in alcohol sales prices was decided as early as the end of the 1960s. At the beginning of the 1980s, a litre of vodka cost the same price as 3 kg of meat or 2 kg of butter, and 20-25 litres was equivalent to the average monthly salary. The perception of the need for in-depth social and economic changes led to the application of more restrictive measures at the beginning of that decade. Drinking establishments were closed and the sale of alcohol was authorized only during certain hours. In spring 1985, the inevitability of reinforcing the social policy led to what has been termed the 'dry law', intended to take these measures even further. However, it met up with the contradiction that the State was in need of money, and the sale of alcohol put plenty in its coffers. This type of law often results in a transfer of these resources from the State to parallel circuits.

The reinforcement of the restrictive measures in 1985 had an evident impact on mortality, which has been demonstrated by cause-of-death analyses (Shkolnikov *et al.*, 1995a): the reduction was essentially in exogenous causes (road and labour accidents, suicides, homicides...). However, as we have seen, this reaction is related to the very specific nature of Russian mortality. Such a sharp impact, on processes resulting from a long deterioration of the relations between society and economy, could not plausibly

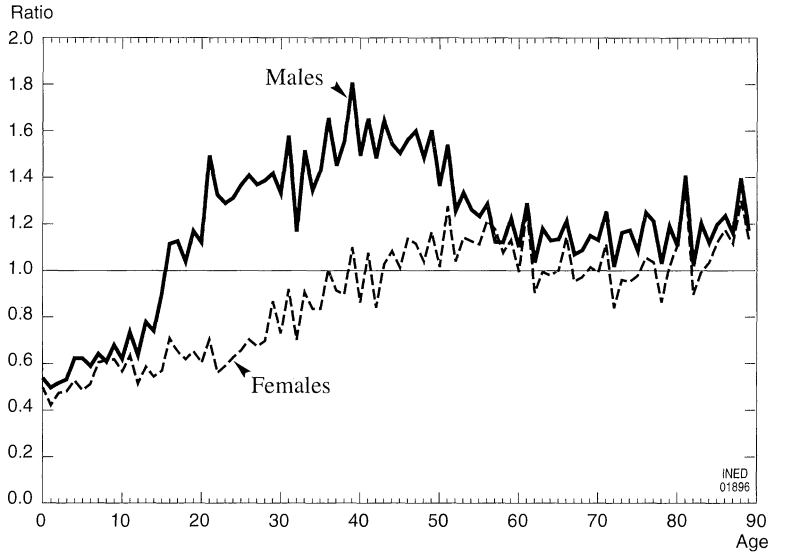


Figure 17. – Mortality change between 1959 and 1980, males and females (ratio of death rates 1980/1959)

Figure 18. – Mortality change between 1980 and 1987, males and females (ratio of death rates 1987/1980)

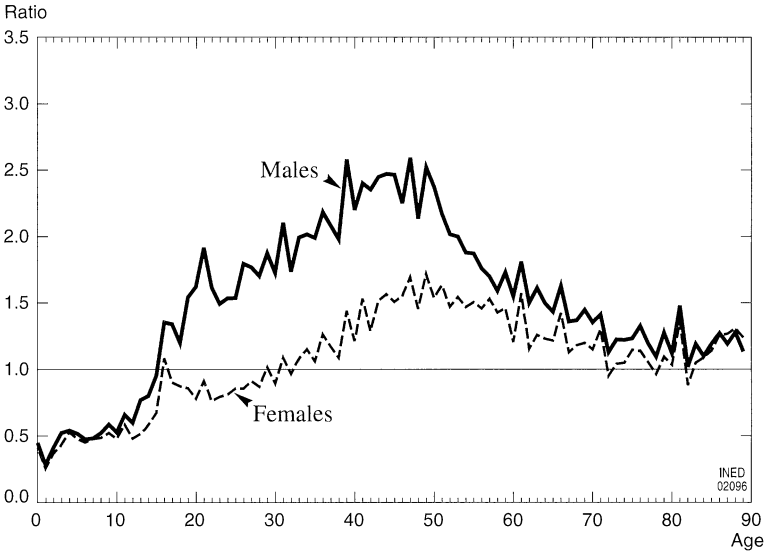


Figure 19. – Mortality change between 1959 and 1994, males and females (ratio of death rates 1994/1959)

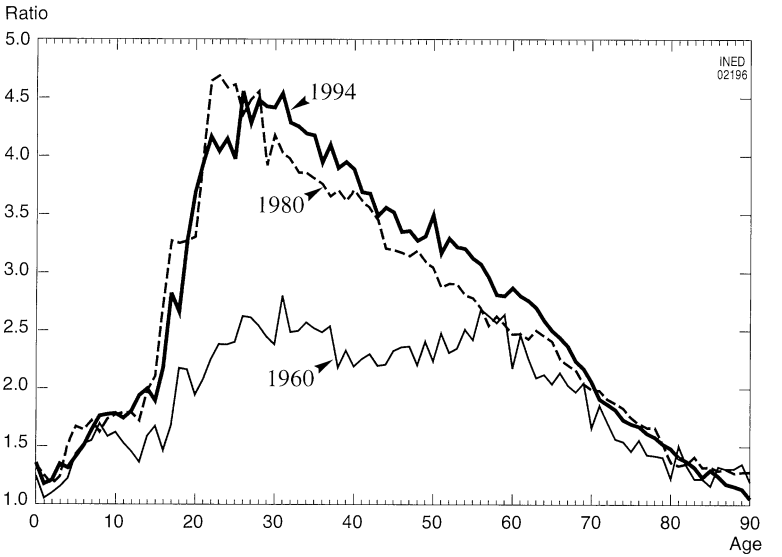


Figure 20. – Excess male mortality in Russia in 1960, 1980 and 1994 (ratio of male to female death rates)

be expected to last. Only a slower evolution will really express the emergence of in-depth changes.

The present study suggests, therefore, that the rise in mortality that began in 1988, and accelerated sharply in 1993 and especially in 1994, was in great part a feedback response to the downturn in the mid-1980s. It cannot be considered simply as a consequence of the changing politico-socio-economic scene. It has not affected the specific nature of Russian mortality (Figure 19), nor the excess male mortality, which displays a similar pattern today to in 1980 (Figure 20).

Conclusion

The object of this study was both theoretical and practical.

The consequences of heterogeneity on population dynamics are illustrated by taking the example of the recent mortality trends in Russia. By introducing the assumption of heterogeneity, we were able to establish a relationship between the short-term and long-term dynamics, which we could not have explained otherwise. Thus, the study suggests a way to investigate more closely the short-term/long-term relationship and the consequences of sudden sharp mortality fluctuations on later trends. This is, of course, hypothesis, but the type of model presented here opens up perspectives that could usefully be explored.

The study shows it is not feasible to consider the sudden mortality variations of the last five years in a vacuum, without also considering the more long-term dynamics. In particular, our model supposes that, under the assumption of heterogeneity, the mortality indicators (life expectancy, probabilities of dying or survivorship) can have deteriorated without there having been any increase in individual risks, compared to 15 years ago. Very short-term structural changes may have produced such variations overall, but not within the different sub-populations. The model proposed here is not the only one that can handle this relationship between long term and short term, and thus explain fluctuations without taking only the contemporary context into account. Other models might suppose there was a real deterioration; but it would in any case be much less acute than the crude data suggest. One might also imagine that a sudden drop in mortality like the one observed in the early 1980s subsequently increased the individual risk of dying among those survivors who would otherwise have died (that is, without the reduction of mortality from exogenous causes). That has never been observed, but nor have mortality fluctuations of this nature ever been observed anywhere in peacetime. There are, of course, other elements that can be investigated. What we hope to have shown here is that it is essential to consider such models before launching into any interpretation of these mortality variations.

Alexandre AVDEEV, Alain BLUM,
Serge ZAKHAROV, Evgeny ANDREEV

Appendix 1
Estimation of cohort mortality

To estimate cohort survivorship probabilities, we had the sex/age structure of the population estimated by the Goskomstat on 1 January of each year from 1959 to 1995 inclusive, and the distribution of deaths by sex and age at death from 1959 to 1994. But we had no distribution of deaths by birth cohort, and this could not be derived from the annual population pyramids because of substantial migration: emigration until 1991, then immigration. In 1959, there was a negative balance of migration amounting to – 1 million, while in 1994 it was almost as strongly positive: + 900,000.

The deaths therefore had to be redistributed by birth cohort and age at death. Such redistribution is not straightforward in the case of Russia, where cohort size may vary considerably in the space of a few cohorts. We thus chose a method very similar to the one described by Noël Bonneuil (Bonneuil, 1994), which consists in interpolating surfaces of deaths cumulated by age and year of occurrence, using a two-dimensional B-spline. We note briefly the principles of this method:

Let $d(a, t)$ be the deaths, at age a on 1 January of year t, occurring during calendar year t;

and $D(a, t)$ the deaths cumulated to age a and year t, that is:

$$D(a,t) = \sum_{u \le a} \sum_{\tau \le t} d(u,\tau)$$

The surface thus obtained is then interpolated using a function that is continuous and can be derived up to a given degree, a sum of polynomes (B-splines)[7]. The $D(\alpha,\tau)$ values can then be calculated for real values of α between a and $a+1$ and of τ between t and $t+1$. Thence we derive the $d(\alpha, \tau)$ values at the same points. These are calculated on 24×24 size grids. They are then summed to calculate the sum of the $d(\alpha, \tau)$ values in the upper and lower triangles of the grid, the diagonal being equally distributed in the two triangles. The deaths are distributed thus by birth cohort and age at death.

Next, the probabilities of dying within a calendar year are calculated in the same way:

denoting $\delta(a, g)$ the deaths in cohort g in year $t - a + 1$, deaths occurring in part at age a and in part at age $a + 1$, the calendar-year probability of dying at age a for cohort g is estimated by:

$$q_a(g) = \frac{\delta(a,g)}{P(a,g) + \dfrac{P(a+1,g) - P(a,g) + \delta(a,g)}{2}}$$

where $P(a, g)$ is the size of the cohort g reaching age a.

[7] We used the computer package BS2VL from IMSL.

Appendix 2
Adjustment of survivorship functions in the absence of disturbance

To extrapolate the survivorship functions in the absence of disturbance, we used the survivorship function of birth cohort 1938 as standard. We denote:

$l(a, g)$ the survivorship function of cohort g, at the age a attained on 1 January of the year $g + a - 1$.

$l_{20}(a,g) = \dfrac{l(a,g)}{l(20,g)}$ survivorship at age a among survivors to age 20 in cohort g.

$$\text{logit}(l_g) = \log\left(\frac{l_{20}(\cdot,g)}{(1 - l(\cdot,g))}\right)$$

We then estimated the α_g and β_g parameters of the regression of logit (l_g) on logit (l_{38}), from the ages reached by cohort g before 1984, that is, $a \leq 1984 - g + 1$. We next estimated the $(l_{20}(a, g))$ logits for any a as:

$$\text{logit}(l_{20}(a,g)) = \alpha_g + \beta_g \text{ logit}(l_{20}(a,1938))$$

and thence $l_{20}(a, g)$ by inverting the logit function thus obtained, in the absence of disturbance.

Appendix 3
Population heterogeneity and mortality change

Let the Russian population be made up of two distinct sub-populations, one with a higher mortality level than the other. To simplify, we shall follow one birth cohort and compare the risk of dying in the absence or presence of a policy to limit the consumption of alcohol.

Let both sub-populations be determined at birth and let their respective proportions at birth of the study cohort be $\alpha(0)$ and $\beta(0) = (1 - \alpha(0))$ or, more simply, α and β. These proportions will naturally vary with age, the survivorship functions being supposed different for each sub-population. We denote $\alpha(a)$ and $\beta(a)$ the proportions at age a.

The first sub-population has a mortality risk $\mu_1(x)$ which varies with age x. The second sub-population is subject to the mortality risk $\mu_1(x)$ and also to an additional risk $\mu_2(x)$.

The survivorship function for the whole population can thus be written:

$$l(a) = \alpha \exp\left(-\int_0^a \mu_1(x)dx\right) + \beta \exp\left(-\int_0^a (\mu_1(x) + \mu_2(x))dx\right)$$

and the risk of dying at age a is:

$$\mu(a) = -l'(a)/l(a) =$$

$$\frac{\mu_1(a)\alpha \exp\left(-\int_0^a \mu_1(x)dx\right) + (\mu_1(a) + (\mu_2(a))\beta \exp\left(-\int_0^a (\mu_1(x) + \mu_2(x))dx\right)}{l(a)}$$

Now let us suppose that between ages a_0 and a_1 an energetic policy to limit alcohol consumption is implemented, and that this policy removes the excess mortality risk $\mu_2(x)$ between these two ages. The second sub-population will be subject only to the risk $\mu_1(x)$ between these ages, but to the risk $\mu_1(x) + \mu_2(x)$ at all other ages.

Thus, the risk $\mu(x)$ does not change up to age a_0. Between ages a_0 and a_1 it falls, and is $\mu_1(x)$ for any x between a_0 and a_1.

The situation changes after a_1. The survivorship function for any $a \leq a_1$ is written:

$$l(a) = \alpha \exp\left(-\int_0^a \mu_1(x)\mathrm{d}x \right)$$

$$+ \beta \exp - \left(\int_0^{a_0} (\mu_1(x) + \mu_2(x))\mathrm{d}x + \int_{a_0}^{a_1} (\mu_1(x))\mathrm{d}x + \int_{a_1}^{a} (\mu_1(x) + \mu_2(x))\mathrm{d}x \right) \text{ or:}$$

$$l(a) = \alpha \exp\left(-\int_0^a \mu_1(x)\mathrm{d}x \right) + \beta \exp\left(-\int_0^a (\mu_1(x) + \mu_2(x))\mathrm{d}x \right) \exp\left(\int_{a_0}^{a_1} \mu_2(x)\mathrm{d}x \right)$$

If we denote $\lambda(a_0, a_1)$ a factor of heterogeneity defined by:

$$\lambda(a_0,a_1) = \exp\left(\int_{a_0}^{a_1} \mu_2(x)\mathrm{d}x \right)$$

and $l_1(a)$ and $l_2(a)$ the survivorship functions associated respectively with the risks $\mu_1(x)$ and $\mu_2(x)$, and noting that the survivorship function of the first sub-population is written $l_A(a) = l_1(a)$ and that of the second sub-population, in the absence of an anti-alcohol campaign, is written $l_B(a) = l_1(a)\ l_2(a)$, then we have:

$$l(a) = \alpha\, l_1(a) + \beta\, l_1(a) l_2(a)\, \lambda(a_0,a_1)$$

Thence the mortality risk at the corresponding age a can be written:

$$-l'(a)/l(a) = -\frac{\alpha\, l'_1(a) + \beta\,(l'_1(a)\, l_2(a) + l_1(a)\, l'_2(a))\, \lambda(a_0,a_1)}{\alpha\, l_1(a) + \beta\, l_1(a)\, l_2(a)\, \lambda(a_0,a_1)}$$

This mortality risk is therefore a function of $\lambda(a_0, a_1)$, and the derivative is expressed by:

$$\frac{\partial \mu}{\partial \lambda} = \frac{-\partial\,(l'(a)/l(a)}{\partial \lambda} = -\frac{\partial}{\partial \lambda}\left(\frac{\alpha\, l'_1(a) + \beta(l'_1(a)\, l_2(a) + l_1(a)\, l'_2(a))\, \lambda(a_0,a_1)}{\alpha\, l_1(a) + \beta\, l_1(a)\, l_2(a)\, \lambda(a_0,a_1)} \right)$$

$$= \frac{[\alpha\, l'_A(a) + \beta\, l'_B(a)\, \lambda(a_0,a_1)]\, \beta\, l_B(a) - \beta\, l_B(a)[\alpha\, l_A(a) + \beta\, l_B(a)\, \lambda(a_0,a_1)]}{\alpha\, l_1(a) + \beta\, l_1(a)\, l_2(a)}$$

$$= \frac{\alpha\beta l_A(a)\, l_B(a)(-\mu_A(a) + \mu_B(a))}{(\alpha\, l_A(a) + \beta\, l_B(a)\lambda(a_0,a_1))^2}$$

$$= \frac{\alpha\beta l_A(a) l_B(a)\mu_2(a)}{(\alpha\, l_A(a) + \beta\, l_B(a)\, \lambda(a_0,a_1))^2}$$

It is thus an increasing function of λ, proportionally to the risk $\mu_2(a)$, the excess mortality risk at age a in the second sub-population.

We note also that the share of the second sub-population, at age $a > a_1$, is written:

$$\beta(a) = \beta\, l_B(a)\, \lambda(a_0,a_1) / (\alpha\, l_A(a) + \beta\, l_B(a)\, \lambda(a_0,a_1))$$

which is also an increasing function of λ.

These equations provide an easy way to simulate this heterogeneity, and above all they clarify the hypotheses made in the article.

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AVDEEV (Alexandre), BLUM (Alain), ZAKHAROV (Serguei), ANDREEV (Evgeny).– **The reaction of a heterogeneous population to perturbation. An interpretative model of mortality trends in Russia**

The rapid increase in mortality in Russia during the past ten years is perplexing. Several authors have explored these movements in depth. In this paper, the authors offer a new interpretation of these dynamics which complements those put forward by previous authors. It shows that the dynamics cannot be studied without taking account of previous movements observed since the 1960s. They suggest that recent developments are the cumulative result of a compensation mechanism which followed the rapid decrease in mortality which occurred around 1985, and of the continuation of a deterioration movement which began in the early 1960s. They do not represent a new trend to a rapidly deteriorating situation. To prove this and to explain these rapid fluctuations, the authors have developed a model based on hypotheses which involve heterogeneous populations. In addition to analysing mortality in Russia, they provide new models which relate to the consequences of heterogeneity on the dynamics of mortality.

AVDEEV (Alexandre), BLUM (Alain), ZAKHAROV (Serge), ANDREEV (Evgenij). – **Réaction d'une population hétérogène à une perturbation. Un modèle d'interprétation des évolutions de mortalité en Russie**

La mortalité en Russie ces dix dernières années a intrigué, par la rapidité de son augmentation. Plusieurs travaux ont d'ores et déjà étudié ces dynamiques de façon approfondie. Cet article offre une interprétation nouvelle de ces dynamiques, qui complète celles qui ont pu être données jusqu alors. Il montre qu'il n'est pas possible d'examiner ces dynamiques sans prendre en compte les évolutions observées depuis les années 1960. Il interprète les récentes évolutions comme la somme d'un mécanisme de rattrapage qui a suivi la baisse rapide de la mortalité des années 1985, et la continuité de la dégradation entamée au début des années 1960. Il ne s'agit donc pas d'une nouvelle tendance à une dégradation accélérée. Pour démontrer cela, les auteurs développent un modèle qui explique la rapidité des fluctuations par des hypothèses sur l'hétérogénéité de la population russe. Ils fournissent ainsi, au-delà de l'analyse de la mortalité en Russie, une nouvelle contribution à la modélisation des conséquences de l'hétérogénéité d'une population sur les dynamiques de mortalité.

AVDEEV (Alexandre), BLUM (Alain), ZAKHAROV (Serge), ANDREEV (Evgenij).– **Reacción de una población heterogenea a una perturbación. Un modelo de interpretación de las evoluciones de la mortalidad en Rusia**

El rápido aumento de la mortalidad en Rusia durante estos últimos diez años ha despertado interés. Varios trabajos han analizado las dinámicas dela mortalidad en profundidad. En este artículo se ofrece una interpretación nueva, que completa las ya existentes, de tales dinámicas. Se demuestra que no es posible examinarlas sin tener en cuenta las evoluciones observadas desde los años sesenta. Las evoluciones recientes se pueden interpretar como la suma de un mecanismo de recuperación que siguió a la rápida disminución de la mortalidad observada desde 1985 y la continuidad de una degradación iniciada a principios de la década de los sesenta. No se trata pues de una nueva tendencia a una degradación acelerada. Para demostrarlo, los autores desarrollan un modelo basado en las hipótesis de poblaciones heterogeneas que explica la rapidez de las fluctuaciones. Su aportación va pues más allá del análisis de la mortalidad en Rusia ; se trata de una nueva contribución a la modelización de las consecuencias de la heterogeneidad sobre las dinámicas de mortalidad.

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